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The Battle of the Conscience

By
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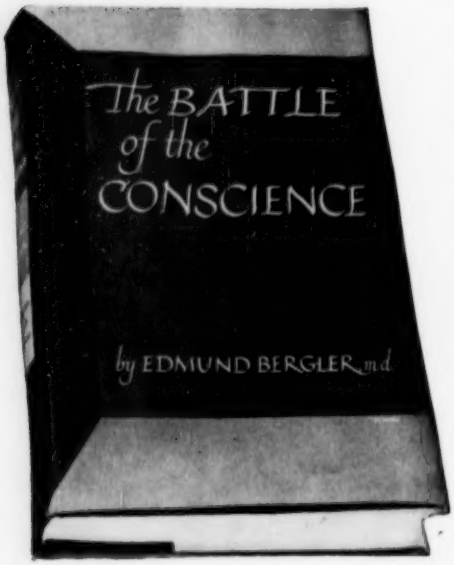

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CONTENTS

PAGE

EARLY AMBULATION AND PERINEAL CARE
Archibald P. Hudgins 69

THE USE AND ABUSE OF TESTOSTERONE THERAPY
Max A. Goldzieher 74

HYPERESTROGENISM TREATED WITH PROLACTIN
Manuel M. Villaverde 84

EFFECT OF ALPHA TOCOPHEROL ON ABNORMAL ELECTROCARDIOGRAPHIC FINDINGS
Arthur Vogelsang 94

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Early Ambulation and Perineal Care

ARCHIBALD P. HUDGINS, M.D.

Few subjects have aroused as much widespread interest in recent years as the problem of early ambulation. It has its vigorous advocates and strong opponents.

Doctor W. J. Dieckmann (Chicago Lying In) recently asked in a discussion, "The reports concerning ambulation are always favorable; but soon most hospitals discontinue the practice without reporting the reason. If it was so wonderful, why has it been given up in the past?"

In an effort to find out what the physicians now think about early ambulation, 12,862 questionnaires were sent to 925 gynecologists and 2071 obstetricians over the United States and to 813 general surgeons and 4746 general practitioners. The last classification was to try to determine how this group compared with the men who were limiting their work to a specialty.

The state of Pennsylvania was taken as a representative segment and the general practitioners and general surgeons were questioned in this area.

A copy of the questionnaire form is shown (Chart I).

CHART I

OBSTETRICAL AND GYNECOLOGICAL STATISTICAL QUESTIONNAIRE

1 *What day do you get your patient up?*

Number of days

After Delivery:	1	2	3	4	5	6	7	8	9	later
With stitches										
Without stitches										
"Normal"										
Complicated										
After Surgery:										
Laparotomy										
Perineal repair										

2 *Early Ambulation*

A. Have you ever used?

Yes No

Still use?

Yes No

B. If Abandoned—Why?

1. Bad results? . . . . . .
2. Patients object? . . . . . .
3. Other reasons . . . . . .

C. Not favored because . . . . . .
Now favored because . . . . . .

3 *What postoperative perineal care do you use?*

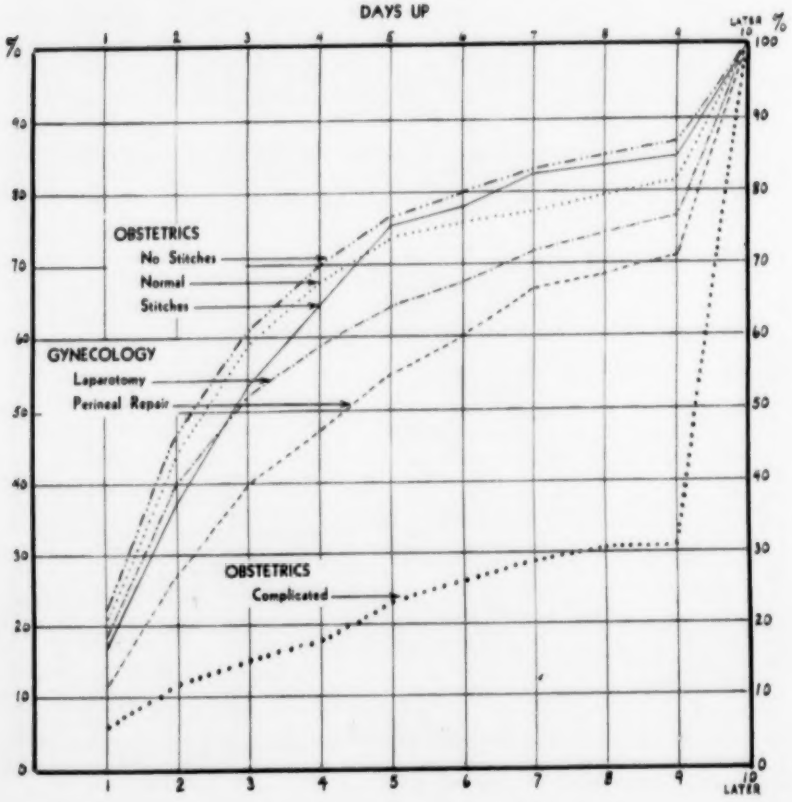
4 *For how many physicains do you speak?* 1-2-3-4

CHART II

FISHER STEVENS SERVICE (MAILING) TO:

Obstetricians (entire U. S. A.)	3071
Gynecologists (entire U. S. A.)	925
Ob-Gyn. (entire U. S. A.)	4307
Surgeons (Pennsylvania only)	813
General Practice (as sample)	4746
Total sent	12802
Answer received	4182
Not in practice, etc.	406
Tabulated	3776–3776

CHART II



The first question was to determine the day the patient was allowed up after delivery—"with stitches," "without stitches," "normal," "complicated," etc.

The graph (Chart III) shows the response to this query.

Approximately 20% of the physicians get the vaginal deliveries and laparotomies up on the first day. Fewer non-obstetrical perineal repairs (14%) and complicated obstetries (5%) are allowed out of bed on the first day.

The out of bed time for vaginal deliveries "with stitches," "no stitches," "normal" and gynecological laparotomies vary but little. Between 54 and 62% of the physicians allow this group up by the third day. Seventy-five to 78% by the fifth day. Of laparotomies 68% are up by the fifth day.

Gynecological perineal repairs are kept in bed longer. Forty per cent are allowed up by the third day and 55% by the fifth day.

The physician's interpretation of the term "complicated obstetries" is perhaps too variable to be of statistical value. The time of arising as shown by the chart is much slower.

The specific question about "Early Ambulation" was repeated for two reasons. Physicians vary in their interpretation of early ambulation from "as soon as the patient reacts from anesthesia" to "three or more days." Furthermore, specific objections to, or arguments for, this method were asked.

The reasons given by the physicians why early ambulation is used or is not used or had been abandoned were grouped within certain broad limits. In listing the discussion for accepting or rejecting early ambulation the ideas centered about the same problem—chiefly patient acceptance, healing, physiology and patient's morale.

Early Ambulation

Favored because:	*Not favored because:*
1. "Patient likes"	1. "Patients object"
2. "Healing better"	2. "Healing poor"
3. "Physiology better"	3. "Not physiological"
4. "Morale improved"	4. "Fear of . . ."
5. "Veins—healthy circulation"	5. Other reasons
6. Other reasons	

Many stated their opinion for or against early ambulation without specifying the reason.

Eighty-eight per cent of the 3,776 tabulated returns stated they had at some time used early ambulation. Ninety-two per cent of these who had used early ambulation are still using it.

Eighty-two per cent of all the physicians responding were still using early ambulation by their statement.

With the hope of getting additional information from this letter,

question number three was added: "What postoperative perineal care do you use?" The question was left entirely open so that the physician may give free expression supporting or criticizing any given method.

The diversified methods of postoperative care were easily grouped under the following subdivisions:

1) Dry, 2) Wet, 3) Antiseptics, 4) Anesthetic, 5) Preparation and whether or not perineal pads were used.

Dry:

1. Method not specified	150
2. Lamp	826
3. Ice	10
4. Other dry methods	58

Wet:

1. Compresses	31
2. Vulva flushes (external)	1531
3. Vaginal douches	20
4. Sitz baths	14
5. Other methods which were grouped under this classification	15

Antiseptics:

1. Merthiolate	308
2. Zephiran	116
3. Mercurochrome	87
4. Sulfas	46
5. Penicillin	437
6. Other medicines not specifically outlined.	503

Anesthetic preparation:

1. Nupercaine	18
2. Surfacaine	17
3. Diothane	0
4. Witch Hazel	22
5. Vaseline	10
6. Glycerine	8
7. Other classifications	5

The subject of perineal pads was expressed specifically pro and con by a few physicians, the numbers being noted in the chart.

Pads:

Yes	59
No	24

The purpose of this paper is not to advocate any specific method of treatment but to present the answers to the questionnaires sent to determine physicians' attitude toward early ambulation and postoperative perineal care.

Conclusions

1. Questionnaires were sent to 12,862 physicians, all U. S. obstetricians and gynecologists and a limited number (those in the state of Pennsylvania) of general practitioners and general surgeons.

2. Answers were received from 4182. Subtracting those "not in practice," etc., the 3776 "active" answers were tabulated.

3. Of the 3776 returns, 3348 (88%) reported early ambulation had been used. Three thousand three hundred forty-eight (92.68%) of those who had used it, still used the method. The 3348 are 82.76% of the total returns who are still using early ambulation.

4. The reasons for abandoning early ambulation were:

a. Bad results	85
b. Patient objects	54
c. Other reasons	106

5. Postoperative care: *Wet* methods were advocated by 1611; *dry* method, by 1044.

Local antiseptics were used by 1497.

Anesthetic preparations were used by 89.

Pads were advocated by 59, opposed by 24.

Professional Building,

Charleston West Virginia,

The Use and Abuse of Testosterone Therapy*

MAX A. GOLDZIEHER, M.D.

Testosterone or as it is also known, the male sex hormone, was used originally for replacement therapy in cases of deficiency of male sex hormone production. This therapeutic purpose is well served in case of hypogonadism due to improper development of the testes accompanied by poor growth of the external genitalia, lack of sexual maturation and absence of secondary sexual characteristics. Administration of testosterone in adequate dosage produces considerable growth of the penis, unfolding of the scrotum and even enlargement and improved secretory capacity of the testicles. Simultaneously, sexual maturation is noted with growth of pubic and axillary hair; the effect of maturation also makes itself felt along mental and emotional lines. The use of testosterone is similarly successful in the male castrate or in the elderly male whose testicular secretion appears to be exhausted; in the latter case, the male climacteric, testosterone therapy offers most gratifying therapeutic experiences to the physician. Less spectacular and often disappointing are the results of treatment in impotence except for specific, well-selected cases in which competent substitution therapy accomplishes its purpose.

While the uses of male sex hormone therapy are generally gratifying in a fairly wide field, they do not exhaust by any means the much more varied possibilities of this pharmacologic agent. As a matter of fact, testosterone can be used to good advantage in a great many other conditions, provided administration is based on sound physiologic principles. Indeed animal experiments, as well as experience in humans, have shown that the so-called male sex hormone evokes numerous metabolic reactions. These effects are so varied and of such magnitude that one is tempted to consider testosterone primarily as a metabolic, and only incidentally, as a sexual hormone.

The metabolic effects of testosterone are particularly prominent in respect to the synthesis of new protein. Formation of new proteins is demonstrated by the positive nitrogen balance accompanied by retention of potassium, sodium, sulphur and water.[1] The anabolic action of testosterone is associated with an elevation of the metabolic rate and is indicative of increased oxygen consumption needed to provide the energy for the synthesis of new body substance. The opposite phenomena, i. e., lessened energy requirements, are noted in the castrate or in marked hormone deficiency with accumulation of fat as the consequence, unless food intake is

*From St. Clare's Hospital, New York, New York.

substantially reduced. The deposit of fat in the castrate or in the elderly male is related to the decreased physical activity, yet the problem appears to be more complicated, for the characteristic distribution of fat depots, at least in homo sapiens, is significant: instead of being universal, as one would expect in overnutrition, the fat accumulates mainly in the abdominal region and is resistant to dietary restrictions.

The synthesis of proteins is the prerequisite of growth, which suggests a similarity between the effects of testosterone and the growth hormone of the pituitary.[2] This similarity, however, is only superficial because production of protein by the growth hormone is practically confined to the period of childhood and early adolescence. Later in life, when longitudinal growth of the skeleton is terminated by fusion of the epiphyses, the growth hormone can produce skeletal growth only about the "akra" while growth of the musculature, though mechanically not limited, is far from conspicuous.

It seems that the task of stimulating growth in the male after pubescence is taken over by testosterone, the effects of which account for the greater muscular development, height and strength of the male as compared with the female of the same breed.

The physiological response to testosterone has been made good use of to speed up retarded growth in adolescents. When we first tried to use testosterone for this purpose (1936), the experiment was considered a risky one because it was believed that the medication might lead to rapid fusion of the epiphyseal lines and thus arrest growth after a short-lived initial spurt.[3] Clinical observation, however, dispelled these misgivings. It is now generally accepted that testosterone does not abnormally speed epiphyseal fusion. This is particularly true in the hypogonad which can be safely treated with substantial doses of the hormone. In case of normal sexual maturity and correspondingly advanced ossification, the effects of the hormone on bony development should be checked by serial x-rays.

Testosterone therapy is highly effective also in the hypopituitary dwarf. The results observed make us question whether the retardation of growth at the time of pubescence is due merely to the lack of pituitary growth hormone or also to a considerable extent to the absence of endogenous testosterone as the result of pituitary gonadotropic deficiency. The correctness of this latter view is supported by the observations that longitudinal growth in such children can be speeded up by substantial doses of chorionic gonadotropin.

The growth effect of testosterone does not seem to be necessarily associated with sexual effects. Female children, given suitable amounts of testosterone either with or without added estrogen, also respond with increased longitudinal growth. Moreover, testosterone has been administered to children at an earlier period of life when sexual effects of the hormone would be unphysiological; yet, the anabolic effects are still obtained even at a relatively low and safe dosage level, with often spectacular increments of body weight and muscular development. Even in premature

infants, administration of testosterone helps to produce gain in weight, apparently by correcting the characteristic disturbances of protein metabolism of prematurity.[4]

The growth promoting effects of testosterone primarily influence the formation of new body substance; hence it stands to reason that the hormone will be specially helpful if anabolism is impaired and the utilization of food valences for the building or maintenance of body substance has become inadequate. Thus, for instance in malnutrition, be it due to poor absorption of food, cachexia, or varied metabolic disorders, testosterone medication elicits a sudden gain in weight. Another excellent example is hypopituitarism or Simmond's cachexia. If a sufficiently potent pituitary extract is not available, attempts at pituitary substitution therapy are bound to fail; administration of testosterone, however, alone or in combination with other hormones secreted under pituitary stimulation, produces remarkable clinical improvement, but especially gain in weight. Similar favorable response is obtained also in patients whose weight loss has been originally caused by severe malnutrition, as for instance, in so-called anorexia nervosa. These patients, in the course of malnutrition, develop a secondary pituitary deficiency and with it, a lack of anabolic hormones, hence even enforced food intake cannot solve their problem. Administration of testosterone breaks the vicious circle; the patient gains weight, the appetite perks up and the emotional situation, as a rule, tends to improve. In Addison's disease, addition of testosterone to the ordinary substitution therapy with cortical hormone or Doca is also useful in increasing muscular strength and vitality. Testosterone therapy in Cushing's disease seems to be less satisfactory than suggested by earlier overenthusiastic reports.[5] At any rate, the theory that Cushing's disease represents excessive adrenal cortical function minus the nitrogen storing effect of androgenic cortical steroids, is not borne out by clinical experience.

We have mentioned the treatment of the male climacteric among the uses of the hormone for sexual purposes. However, such a simplification is hardly correct because most of the symptoms of the male climacteric are independent of the sexual aspects, and apparently linked to the metabolic activities of the hormone. Libido and sexual power are not necessarily abolished in the male climacteric and definite symptoms may be forthcoming without noticeable decline of sexual activity. The theoretical evidence is also relevant here. Thus, increased urinary excretion of the follicle-stimulating hormone is no evidence that the Leydig cells are secreting less testosterone for they are under the influence of the interstitial cell-stimulating hormone and independent of the follicle-stimulating hormone. This relationship is shown also by the inability of testosterone to suppress the secretory activity of the pituitary, even if given in substantial doses.[6]

The most important symptoms of the male climacteric—vasomotor disturbances, headache, insomnia and emotional or mental manifestations—are well controlled by adequate hormone therapy, even though in many

cases, the administration of testosterone has but little effect upon the impotence.

The metabolic benefits of testosterone therapy are particularly conspicuous in the elderly or senescent patient. Aging tissues are characterized morphologically by the smaller size of the cells and their nuclei; this is in accord with their decreased water content and the slower replacement of cytoplasm. Upon the impact of testosterone, the cells increase the rate of exchange of their worn-out constituents; their water content is augmented and they revert to the morphology of normally active, not senescent cells. This could be clearly demonstrated in our experiments in which testosterone was applied directly to the atrophic skin of aged individuals. Testosterone, irrespective of the sex of the patient, changed the atrophic epidermis to a tissue morphologically similar to that of a normal presenescent skin.[7] We are engaged, at present, in spectrophotometric studies on light reflected from the skin to confirm that the morphological changes elicited by testosterone are accompanied, or even preceded, by biochemical changes such as an increased ribonucleic acid content of the epidermal cells.

The effectiveness of testosterone in the senescent individual is not limited to the skin; this is clearly shown by the weight increase of such patients. This gain in weight does not necessarily express the result of increased food intake, for it occurs also without increased eating. It seems to depend primarily on two factors: retention of water and a positive nitrogen balance indicative of the building up of new cytoplasm. Retention of other substances used for the building of body tissues is concomitant; particularly noteworthy is the retention of calcium salts, for example in cases of marked senile osteoporosis. In patients who have been bedridden or whose extremities have been immobilized for a long period, osteoporosis develops in the course of inactivity. Both the osteoporosis of senility and of inactivity are successfully controlled by administration of testosterone, not only in the male but also in the female. In the latter, it is evidently advisable to combine testosterone with suitable quantities of estrogen, meant to take away the "androgenic" edge of the medication and to prevent undesirable virilization without diminishing the metabolic effect of the hormone. Startling calcium retention and recalcification of the skeleton has been obtained with such combined estrogen-testosterone therapy in osteoporotic aged females.

Combination with estrogen seems advisable also if testosterone therapy is aimed at suppression of excessive pituitary activity, for testosterone is a far inferior inhibitor of the pituitary than the female sex hormone. Thus, in thyrotropic exophthalmos, that is, when the protrusion of the eyeballs is referable to excessive secretion of thyrotropic hormone in the course of thyroid disease, testosterone therapy alone, even in fairly large doses, is rather ineffectual. Addition of estrogen, however, changes the response completely, curbs the pituitary hypersecretion and thus, arrests the protrusion of the eyeballs. This is accomplished without any of the untoward alterations estrogen therapy might otherwise elicit in the male. Hence the

purpose of the combined therapy is the opposite of that proposed for the senile woman; instead of taking the "androgenic edge" away with estrogen, we are removing the "estrogenic edge" by adding androgen.

Discussion of ophthalmological problems has directed our attention to two other, more or less rare conditions which can be benefited by the use of testosterone. One of these is keratoconjunctivitis sicca or Sjoegren's disease, a condition which, though much more common in women of menopausal age, has also been observed in the male.[8] Treatment with testosterone in such cases produces the same beneficial results as administration of estrogen in the female: lacrimal secretion is improved and the changes of the conjunctival and corneal epithelium are corrected. A much less uncommon condition is keratoconus, a disease in which the cornea as well as the sclera are thinned out. This is apparently not due to an abnormally high intra-ocular pressure but seems to be caused by a weakness of the fibrillar structures of the eyeball which are incapable of resisting the normal intra-ocular pressure. In view of its anabolic effect, it was fair to assume that testosterone would strengthen collagenous fibrils just as it increases the bulk and strength of muscle fibrils; hence we embarked on treating such patients with testosterone, with added dietary measures such as low salt — low liquid intake in order to decrease intra-ocular pressure. In a not inconsiderable number of cases so treated, we succeeded in completely arresting the progress of the disease while in some cases the checkup by the ophthalmologist actually showed improvement of the patient's condition.

It may be asked whether the anti-estrogenic effect of testosterone should be classified with the sexual or non-sexual functions of the hormone. In our opinion, the anti-estrogenic effect is a rather complicated one: it consists partly of a depression of endogenous estrogen production both in the male and in the female and partly of a direct action on the target organs inhibiting or counteracting the effect of circulating estrogen. It is obvious that the anti-estrogenic effect of testosterone finds a particularly wide field in gynecological therapy. Testosterone has been used for quite some time to curb menorrhagia and has proved itself successful in the hands of many gynecologists[9] notwithstanding the vehement denunciations of others who consider male sex hormone therapy in the female irrational and "unphysiological." The term, "unphysiological," of course, is unjustified, for androgens are secreted in the body of the female and possibly play a role in the mechanism of the menstrual process. A more justified objection is based on the fact that control of menorrhagia occasionally calls for dangerously large doses of testosterone whereas other methods are available which do not involve the risk of masculinization. However, androgen used with discretion at the right time and in suitable dosage is useful in combating menorrhagia. It can be recommended in selected cases (especially in blond women and in the absence of hypertrichosis). Injections of 25 mg. should be given daily or every other day right after the onset of menstrual bleeding without exceeding a total of 150 mg. per month. It serves no good

purpose to continue treatment during the second half of the cycle. Anti-estrogenic therapy during the lutein phase of the cycle, on the other hand, is useful in case of painful or lumpy breasts usually complained of premenstrually or sometimes during the whole second half of the cycle. Mastodynia with or without cyst formation responds well to testosterone alone or preferably in combination with progesterone which can be given by injection or in the form of sublingual or buccal tablets.

An attempt to control endometriosis hormonally is also justified although our personal experience with this phase of anti-estrogenic therapy is much too limited to permit conclusions. We are definitely convinced, however, of the value of anti-estrogenic therapy in case of uterine fibroids with or without menorrhagia. On the basis of the work of Lipschutz and his group, it seems definitely established that testosterone is anti-fibromatogenic, in other words, counteracts estrogen in the formation of fibroid tumors. It was also shown experimentally that a combination of various steroids is more effective than any single steroid. Our clinical observation has convinced us that testosterone and progesterone combined offer an effective type of non-surgical treatment for growing uterine fibroids. Not every fibromyoma, of course, can be made to shrink; some uterine tumors, however, shrink to such an extent that they are no longer demonstrable by gynecological examination.

The anti-estrogenic effect of testosterone is of value also in the treatment of malignant tumors which derive from the mammary gland or from the uterine mucosa, i. e., from tissues the growth and function of which requires stimulation by estrogen. The beneficial effect of castration on the progress of mammary or uterine tumors is corroborating evidence. Administration of testosterone, especially in inoperable or metastatic mammary cancer, produces effects of considerable significance.[19] No claim is made that breast carcinoma can be cured by hormones, yet it is well established that testosterone is a most desirable palliative, particularly in case of widespread bone metastases. The suffering of these unfortunate patients is hardly controllable by substantial doses of morphine or other analgesics. Testosterone not only decreases or abolishes pain, but sometimes arrests further growth of the skeletal metastases. X-rays as well as biopsies have shown resorption of the metastases and restoration of bone tissue in the place of previous destructive lesions. Unfortunately, testosterone is much less effective and often entirely unsuccessful in curbing the growth of metastases in soft tissues such as lymph nodes liver or lungs. Such failures provoked the experimental use of stilbestrol in large doses, especially in aged patients, for women in the sixties or older seem to do much better on stilbestrol than on testosterone. It is not so surprising, after all, that stilbestrol, a potent estrogen, should have such an effect, in view of the known bi-phasic action of the hormone; in large doses, stilbestrol acts differently than in small doses, for instance the latter are useful to elicit menstrual bleeding whereas large doses can be used most effectively for the

treatment of menorrhagia. Less experience has accumulated concerning uterine tumors; nevertheless, inoperable patients who do not respond to radiation should be given the benefit of hormone therapy.

An interesting aspect of our subject is the effect of testosterone in frigidity of the female. Testosterone has been used for the purpose of restoring libido in the male in the sense of true substitution therapy; the same procedure in the female could hardly be interpreted in the same sense. The treatment with "male sex hormone" was developed empirically when women given testosterone for menorrhagia or other purposes reacted with an increased libido. While the results of systemic treatment by injection or by mouth were quite satisfactory at first, continued treatment seemed ill-advised. The growth of hair on the face and other traits of masculinization were discouraging, and it seemed likely that these physical changes might prove to be emotionally so upsetting that they would suppress libido rather than improve the sexual situation. We have, however, evolved a technic which permits the successful use of the hormone without producing virilization. The technic consists of topical application in ointment form to the clitoris and its immediate vicinity. The noticeable enlargement, hyperemia, and greater erigibility of the clitoris probably play an important role in the favorable response of these patients, for these changes permit easier mechanical stimulation during the sexual act and enhance the irritability of this organ. Whether or not other factors also enter to determine the favorable therapeutic results is still uncertain.

The manifold uses of testosterone for therapeutic purposes make it quite obvious that abuses in the hands of the less well-informed practitioner must be expected. Thus excessive dosage of testosterone has been given to young boys in order to correct sexual or general somatic retardation. It is not so much the premature genital development which may be regretted, but rather the conflict which arises from the maturation of the genitalia in a mentally immature child whose awakening sex urge cannot be in harmony with the coexisting emotional immaturity. Premature sexual development is undesirable in boys below the age of pubescence; nor is sexual overstimulation desirable even after puberty as long as the boy is too young to safely gratify his sexual impulses.

Another pitfall of overdosage in the adolescent is the precipitation of epiphyseal closure. The danger is greater if sexual maturity is farther advanced, and less in the more immature boy, yet it is a fallacy to assume that the same dose of testosterone produces epiphyseal maturation equally in all patients. In view of the many as yet unknown contributory factors, there should be frequent check-ups on the progress of epiphyseal ossification by serial x-rays, and treatment should be stopped as soon as the bone age begins to approach or exceed the chronological age of the patient.

Another, much less valid objection has been raised against testosterone therapy in the adolescent or adult male. It has been claimed that testosterone is injurious to the sperm producing parenchyma of the testes, probably by suppressing pituitary gonadotrophic secretion. The fear of causing

testicular damage is justified neither on the basis of clinical experience nor on theoretical grounds. It has been noted by careful observers that testosterone does not damage the testicles but actually increases their size and functional capacity. Even weightier than the clinical evidence are recent experimental data according to which testosterone at varied dosage levels hardly diminishes and certainly does not suppress excretion of gonadotropins in the urine. Thus the concept must be abandoned that small doses of testosterone, by suppressing pituitary function, impair the testicles, whereas large doses maintain the testes by their direct effect on the testicular parenchyma. A further argument against this view is the fact that the urinary gonadotropins in the hypogonad are of the "follicle stimulating" (FSH) type which does not influence testosterone production; the Leydig cells secrete testosterone under the influence of a different type of gonadotropin (I. C. S. H.) which is not found elevated in the urine of the hypogonad. Moreover, the Leydig cells respond with increased testosterone secretion to chorionic gonadotropin with subsequent growth and improved functional capacity of the spermatogenic tissue.

Of special significance is the abuse of testosterone in women irrespective of the purpose of the therapy. Excessive dosage obviously produces facial and bodily hair growth, deepening of the voice, and occasionally changes of personality; it may even cause lasting impairment of the ovaries and procreative ability. Hence, dosage should be controlled carefully within strict limits and reserved for that period of the cycle during which the most desirable effect can be obtained.

Contraindications exist also in the adult male. First of all, there is the elderly male who responds well in the beginning to testosterone medication, showing greater vigor and a gain in weight. Upon continued treatment, however, the rising weight curve flattens out and reaches a plateau with a subsequent trend downward. Upon continuation of the therapy, urinary ketosteroid excretion decreases as a sign that adrenocortical function has been depressed; thus, excessive dosage of testosterone may actually become harmful to the elderly patient, although these by-effects are preventable by administration of cortical extract. In response to the latter, patients notice further weight gain and a heightened sense of well-being.

Neither hypertension nor hypotension contraindicate the use of testosterone, particularly in middle-aged men, for therapy is followed by a change of blood pressure in the desired direction: by a decrease of blood pressure in hypertension and by an increase of blood pressure in hypotension. It is not difficult to explain this apparently paradoxical reaction: the increase in blood pressure is frequently referable to a state of nervous tension which is likely to decrease if the testosterone requirements of the body are met; a decline of the elevated systolic pressure is concomitant. In the hypotensive patient, on the other hand, hormone therapy causes increased vigor and better muscular tone as the result of which the blood pressure may rise towards a normal level.

Symptoms of angina or coronary sclerosis are not contraindications

to testosterone therapy. Though actual changes of the arteries cannot be reversed by the hormone, the effects upon the nervous system together with some vasodilation may contribute to clinical improvement. In addition to the more ample perfusion of the coronary system, the nutritional status of the myocardium may profit directly by the anabolic effect of the hormone. Notwithstanding the obvious value of testosterone therapy in the aged male or in patients with coronary disease, attention must be called to an element of real danger: some of these patients who were used to a state of invalidism, experience in the course of treatment a replacement of their sense of futility by feelings of vigor and ambition, hence they are tempted to embark on activities which exceed the capacity of their cardiovascular system. The elderly patient who presents signs of organic cardiac disease or coronary changes, should not receive testosterone without being enjoined to remain mindful of the limitations of activity imposed upon him by his cardiac status.

Under the heading of abuses, treatment of cases of prostatic involvement figures importantly. The dependence of the prostate upon stimulation by testosterone is well established. Thus, in the early castrate or hypogonad, the prostate remains undeveloped whereas sufficiently energetic medication achieves satisfactory growth of this glandular organ. Regression of the prostate can be brought about by estrogen, as has been shown in cases of prostatic hypertrophy or cancer. There are, however, elderly patients who complain about bladder symptoms such as frequency, nocturia, dysuria, etc., and whose prostate is not enlarged. Their symptoms are referable to a weakness of the bladder musculature and to a state of nervous irritability which is such a common manifestation of the male climacteric. Upon administration of testosterone, the bladder musculature is strengthened, the nervous irritability subsides and the complaints are either eliminated or at least considerably mitigated. Such patients respond rather well to hormone therapy without demonstrable adverse effects upon their prostate. However, in case of mechanical obstruction of the bladder neck and particularly in the presence of a median lobe, administration of testosterone must be frowned upon, for even a slight further enlargement of the prostate must aggravate the condition, irrespective of any simultaneous improvement in the tone of the bladder musculature. Hence, testosterone therapy must be limited to cases of prostatism without sizable enlargement of the prostate or obstruction of the bladder neck, whearas the real prostatic hypertrophy calls for suitable urological measures or the administration of estrogen.

Our cursory review of the purposes the so-called male sex hormone can be used for, clearly shows that hormone therapy is applicable to a far greater number of non-sexual than sexual conditions. Testosterone, in this respect, does not differ from the majority of other hormones which can be used for many purposes besides mere substitution of an endogenous deficiency. The rapid progress of endocrinology during the past twenty years has success-

fully reduced the indiscriminate and purely empirical use of hormones, so that it had become customary to think of hormone therapy as strictly limited to the substitution of hormone deficiencies. With increased knowledge of endocrine pharmacology, however, it has become apparent that hormones may be used for other than substitutional purposes. Thus, for example, the surgeon gives adrenalin in case of local anesthesia, not because the adrenalin secretion of the patient is deficient but because adrenalin produces local anemia and thus prevents too rapid removal of the locally injected anesthetic. Another example is the use of insulin in psychiatry to produce convulsions and shock, not for the purpose of compensating for endogenous insulin deficiency. Thus these hormones are used as pharmacologic agents in order to produce specific and often altogether unphysiologic reactions. We have pointed out recently how well estrogens are applicable for pharmacologic purposes in a great variety of conditions not within the sexual sphere. Testosterone is not far behind estrogen in this respect, and the physician who is familiar with the pharmacological properties of the hormone possesses a useful addition to his therapeutic armamentarium.

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Hyperestrogenism Treated with Prolactin

MANUEL M. VILLAVERDE, M.D.

GENERAL CONSIDERATIONS ON HYPERESTROGENISM

If we agree with Varangot,[34] hyperestrogenism is a syndrome as clearly evident and as sufficiently proved as hyperthyroidism. Its general characteristics are:

1. Precocious or normal puberty.
2. Menstrual disorders, namely, amenorrhea, hypomenorrhea, hypermenorrhea, or anisomenorrhea. Their common characteristic is a proliferative hyperplasia of the endometrium, that sometimes develops into cystic glandular hyperplasia.
3. Functional bleeding is generally considered a hyperestrogenic disorder, although Abarbanel[1] says that it may proceed from every known type of endometrium. Dysmenorrhea[32] is present in almost all instances.
4. Premenstrual tension, according to Frank,[10] shows hyperestrogenemia and hyperestrogenuria. Israel[17] also found hyperestrogenism in cases of premenstrual tension and midterm bleeding. Midterm tension is a similar occurrence.
5. Congestion of breasts and pelvis, sometimes chronic mastitis: Atkins,[2] Villaverde.[35] Leucorrhea, frigidity (or nymphomania), and sterility due to a permanent proliferative endometrium,[5, 32] are common.

Some other symptoms may also be found, such as toxicosis, gravidarum, fibromyoma of the uterus, fever, epileptic-like seizures and vulvitis associated with stomatitis.

Hyperestrogenism is present in a great number of neuroses and psychoses;[3] and I found [36, 37] hyperestrogenism as a frequent cause of hypoparathyroid irritability.

All these symptoms, particularly when they appear by crises, are associated with high peaks of blood estrogen concentration. Salaber and Del Castillo[29] use estrogens as a test in these cases; they impair the symptomatology. This hyperestrogenism may be either absolute hyperestrogenism or relative, depending on a lack of progesterone.

Diagnosis may be made either by clinical examination[11] or by laboratory tests. Some[4] emphasize the use of biopsy of the endometrium; others[25], the use of vaginal smears. Hall[14] considers vaginal smear the most accurate clinical test for determining the estrogenic levels, and Rubinstein[27, 29] considers the smears as a guide to the condition of the endometrium. Thus diagnosis can be made by carefully studying the symptoms, and verified by biopsy or vaginal smear test.

Following our discussion it would seem that all of the above clinical pictures may be regarded as aspects of one syndrome—hyperestrogenism.

The Use of Prolactin in the Treatment of Hyperestrogenin Syndromes

Hall[15, 16] announced the success of the lactogenic hormone of the pituitary gland in arresting functional uterine bleeding; and Goldzieher[12] found that the hormone is useful in several other conditions also characterized by hyperestrogenism, where its anti-gonadotrophic property may be helpful.[26] Evans, Simpson, and Turpeinen[6] stated that this hormone is luteotrophic, and this was corroborated by other workers.[7, 8, 9] The blood of lactating women has similar effects. It is able to suppress uterine bleeding, and this action might well be ascribed to the presence of prolactin in the blood stream at this time.[20]

Summarizing: the lactogenic hormone of the hypophysis is anti-estrogenic and luteotrophic, and according to Kupperman et al.[19] it produces a proper hormonal balance.

Materials and Methods

For treatment with lactogenic hormone Physolactin, Glaxo and Prolactin (Armour), were used. Forty-two cases were selected, which corresponded to the symptomalogy formerly given above, because their clinical symptomatology can be related to the hyperestrogenic syndrome. Furthermore, the vaginal smears always confirmed the clinical diagnosis of hyperestrogenism. The vaginal smear is indicated in the table as follows: + + + when there is an intense cornification showing high levels of estrogens in blood; + + when the smear corresponds to a normal premenstrual picture. All the smears were taken during the last days of the third week, or during the first days of the fourth, i. e., from about the 18th to the 22nd day of the cycle. The results were given after a comparative study of the smears stained with both the differential stain of Shorr[21] and Mack's iodine vapor stain.[21]

In the cases of uterine bleeding, the patients were injected during the menses; all the other cases were injected during the intermenstrual period. Injections began some days prior to ovulation and were continued for ten or fifteen days. Patients injected for 21 days received the injections practically during the whole intermenstrual cycle. It is easy to calculate the daily amount injected: patients who received 2700 units in 15 days, were injected at the rate of 180 units daily. The doses ranged from 720 units given in 12 days, to 2700 units given in 15, 18, or 21 days; sometimes smaller amounts were given. Treatment was prolonged from 1 to 7 months, one month being the time for one course of injections. In most instances, the lactogenic hormone was injected without simultaneous use of any other

hormone; sometimes testosterone ("T," in the table), or progesterone (P) were added. *Testosterone was given, the proper precautions being taken,[33] during the days preceding ovulation; progesterone, during the days after it. Ovulation was assumed to be 14 days before the expected menstruation.

In the table, the negative sign (−) means no improvement; one plus sign (+) means a fair result; two plus signs (+ +) mean a very good or excellent improvement.

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*Testosterone proprionate: Schering's *Oreton* and C. W. Frost's *Orchisterone*. Progesterone: Schering's *Proluton*.

TABLE

Patient	*Age of patient*	*Vaginal smear*	*Units expressing the amount of lactogenic hormone injected*	*Days*	*Other hormones*	*Basal metabolic rate*	*Comments*	*Symptoms*	*Results*
1	33		1800	10			Menses improved	Sterility Mastodynia Uterine hypoplasia	+
			1800	14		+16	No improvement		
2	34		900	4		+ 1	Worse	Premenstrual tension	+ +
			2700	15	P		Improvement		
3	45		2700	15		+18 5	Excellent (Other treatments had failed before)	Premenstrual tension, anxiety, fibromyoma, hyperthyroidis	+ +
			1800	15			Excellent		
			900	15			Good		
			1800	13			Excellent		
			900	15			Good		
			2700	15		— 6	Excellent		
4	33	+++	1800	15			Delayed menses	Scanty menses, leucorrhea, anxiety	+ +
			900	15		—19	Delayed menses Progesterone had failed		
5	27		1800	15			Improvement	Premenstrual tension Psychic symptoms	+ +
			2100	10			Improvement		
			2400	8	T		Improvement		
6	26		2700	15			Much better than progesterone, which had proved very good	Premenstrual tension, mastodynia, manic depression	+ +
			2700	15					
7	45		2700	12	T P		No improvement	Mastodynia, anxiety	—
8	33		1600	10	T P		Slight improvement	Schizophrenia Premenstrual tension	+
9	18		1800	10		— 1 5	Improvement	Dysmenorrhea. Convulsive state	+ +
			1800	10			Excellent		
			2700	15			Excellent		
			1800	15			Excellent		
			1800	15			Excellent		
10	43		1800	10	P	+ 8	Progesterone proved to be very effective; the addition of lactogenic hormone lessened the amount necessary of progesterone	Premenstrual tension. Manic depression	+ +
			900	10	P				
11	28		2700	18	T P		Improvement	Compulsive state. Premenstrual tension	+ +
			2700	18	T P		Improvement		

TABLE

Patient	*Age of patient*	*Vaginal smear*	*Units expressing the amount of lactogenic hormone injected*	*Days*	*Other hormones*	*Basal metabolic rate*	*Comments*	*Symptoms*	*Results*
12	25		1800	10	T P		Improvement (progesterone	Premenstrual tension,	++
			1800	10	T P		Improvement was very	mastodynia, membra-	
			1800	10	T P		Improvement effective)	nous dysmenorrhea, compul-	
			1800	10	T P		Improvement	sive state	
			900	15	T P		Slight improvement (vago-		
							tonia)		
			1800	15			Improvement, (particularly the		
			1800	15	P		Improvement compulsive		
							state)		
13	31		1800	10			Failure	Membranous dysmenor-	−
								rhea, ovulatory syn-	
								drome	
14	39		2700	15	P	− 1	Improved	Dysmenorrhea	+
			1800	10			Produced amenorrhea		
15	37	+++	2700	15			Excellent	Membranous dysmasto-	++
			2700	15	P		Excellent	dynia	
16	34		1800	12		+ 6	Excellent	Membranous dysmasto-	++
			1800	12			Excellent, and persistent	dynia ovulatory dis-	
							improvement	charge	
17	30	++	1800	15		− 9	Improvement	Membranous discharge	++
			900	15			Improvement		
18	14		2700	15			Excellent	Premenstrual tension,	++
			2700	15			Excellent	mastodynia. Puberty	
								at 11	
19	18		1800	15		− 3	Excellent	Premenstrual tension,	++
								ovulatory tension,	
								mastodynia	
20	33		1800	10		+ 7	Slight improvement	Premenstrual tension,	++
			1800	10			Excellent (cyst disappeared)	chronic mastitis (cystic)	
			1800	10			Improvement		
21	15		1800	10			Excellent	Premenstrual tension	++

22	36	+++	♀700	21		− 6	Failure	Premenstrual tension	+
			♀700	21	P		Improvement		
			♀700	21	P		Failure		
23	25		1800	10	T	+ 9 5	Slight improvement	Premenstrual tension frigidity	+
24	26		♀700	15		−10	Failure	Premenstrual tension; mastodynia, frigidity	−
25	42		900	7			Failure	Premenstrual tension; mastodynia	−
			900	15			Failure		
26	38		1800	7	P		Excellent	Premenstrual tension; mastodynia	++
			1800	7	P		Excellent		
27	30		1800	10	P	+ 3	Worse	Premenstrual tension; frigidity	−
			1800	10			Worse		
28	29	+++	♀700	15	P		Slight improvement	Premenstrual tension; frigidity	++
			720	12	P		Slight improvement		
			720	12	P		Slight improvement		
		+++	♀700	15	P		Excellent		
29	32		1350	15		−10	Improvement	Premenstrual tension; mastodynia	++
			900	15			No improvement		
30	34		960	4			Worse	Premenstrual tension; sterility	++
			♀700	15	P		Improvement		
31	34	+++	1800	15		+25	Improved, particularly mastodynia	Premenstrual tension; mastodynia; uterine bleeding	+
			♀700	15			Nervousness		
			1600	16			Nervousness		
32	45		1800	10			Improvement (amenorrhea)	Migraine ovulatory and premenstrual	++
			1800	10			The amenorrhea continues		
			1800	10			Excellent		
			1800	10			Excellent (amenorrhea)		
			1800	18	P		Migraine. Lactogenic Hormones are her best remedy		
33	36		♀700	15		−13	Excellent	Chronic mastitis	++
			♀700	15			Excellent		
			♀700	15			Excellent		

TABLE

Patient	*Age of patient*	*Vaginal smear*	*Units expressing the amount of lactogenic hormones injected*	*Days*	*Other hormones*	*Basal metabolic rate*	*Comments*	*Symptoms*	*Results*
34	23		2700 2080 2700	21 13 15	P T	− 5	No improvement Better menses No improvement	Chronic mastitis	−
35	33		1800 1800	13 13			Worse Worse	Chronic mastitis, inversion of nipple. (Negative of cancer)	−
36	33	++	1600	10		−20 −11.5	Improvement	Frigidity	++
37	34	+++	900 1800	5 10	P	+ 3	Frigidity improved Amenorrhea Progesterone seems to act much better	Frigidity Amenorrhea	+
38	31		900	5		−11	Some improvement	Oligomenorrhea	+
39	20	+++	1260 1800 1800	6 10 10	 P 	− 3	Arrested bleeding Produced menses after 3 month's absence Normal menses Testosterone and progesterone failed during two months of treatment	Uterine bleeding	++
40	32	+++	2700 2700	15 15	T P P	+ 3	Improvement Excellent	Puberty at 11. Goiter. Nervousness	++
41	36	*	2700	15	P	0	No improvement	Puberty at 11. Nervousness	−
42	15		1800	13		− 2	Failure to improve general state	Puberty at 11	−

* This case had normal androgens in both blood and urine.

NOTE: Three cases were added to this table after computation, and are not estimated in the final results. Two of them failed to improve, and the other one improved poorly.

Results and Comments

The general results of this therapy has been very favorable; only 21.5% of the cases failed to improve:

	Number	*Per cent*
Total cases	42	
Good results	25	59.52
Fair results	8	19.04
No improvement	9	21.44

We can find 10 different symptoms prevailing among the other symptoms of hyperestrogenism. Our patients show one or more of these symptoms, which are tabulated according to their incidence and the results of therapy:

	No.	*Bad*	*Fair*	*Good and excellent*
Premenstrual tension	22	3 (13.63%)	4 (18.18%)	15 (68.19%)
Mastodynia	17	5 (29.4%)	2 (11.8%)	10 (58.8%)
Nervous and Mental Symptoms	12	2 (16.66%)	1 (8.33%)	9 (75.1%)
Dysmenorrhea (membranous, 5)	7	1 (14.28%)	1 (14.28%)	5 (71.44%)
Frigidity	6	2 (33.33%)	2 (33.33%)	2 (33.33%)
Midterm symptoms	3	1 (33.33%)		2 (66.66%)
Uterine bleeding	2		1 (50%)	1 (50%)
Oligomenorrhea	2		1 (50%)	1 (50%)
Migraine	1			1 (100%)
Leucorrhea	1			1 (100%)

Nervous and mental symptoms, and premenstrual tension reacted very well to lactogenic hormone; only in few instances were these symptoms not ameliorated. Mastodynia did not react as well, but, generally speaking, the cases prove that the lactogenic hormone has an excellent action upon these symptoms due to hyperestrogenism. Both cases of migraine and leucorrhea recovered impressively, particularly migraine, which was troublesome during the days of ovulation and those preceding the menses.

There is no relation in regard to age or to basal metabolic rate. Basal metabolic rate is normal in most cases.

The use of testosterone or progesterone did not introduce any fundamental change into the general results of lactogenic hormone therapy.

Summary

In this paper hyperestrogenism is considered an actual clinical entity, and the hyperestrogenic is defined in terms generally agreed on. Diagnosis of the condition may be accomplished either by clinical examination or laboratory tests (biopsy of the endometrium, and vaginal smears, both of them of equal accuracy). Forty-two cases are studied; they correspond strictly to the picture of hyperestrogenism, as demonstrated by means of vaginal smears stained with Shorr's single differential stain, and Mack's iodine vapor stain.

These cases were treated with lactogenic hormone, due to its promising properties as an anti-estrogenic, luteotrophic, regulator of hormone balance. The patients were injected in monthly courses, varying from one to seven, according to the circumstances; the total dosage ranged from 720 units given in 12 days, to 2700 units given in 15, 18, or 21 days, for each course.

Good results were obtained in 25 cases (59.5%); fair, in 8 (19%); and there was no improvement in 9 (21.5%).

Nervous and mental symptoms, and premenstrual tension reacted very well to lactogenic hormone. Migraine and leucorrhea, particularly the former, recovered impressively.

Thus the lactogenic hormone of the pituitary gland seems an effective means of controlling hyperestrogenism, whatever its clinical picture may be.

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Effect of Alpha Tocopherol on Abnormal Electrocardiographic Findings

Arthur Vogelsang, B.A., M.D.

Introduction

It is the purpose of this paper to demonstrate what changes have been observed in various types of electrocardiograms of cardiac patients given vitamin E. All other circumstances being equal, it is the assumption of the author that these changes were caused by the administration of adequate amounts of alpha tocopherol (vitamin E).

In the following cases the dosage of Vitamin E is stated as milligrams per day of alpha tocopherol (bio-assay). Those cases in which digitoxin had been used are excluded, as are those whose degree of exercise had been reduced as part of the treatment.

Data

In those cases which show a P-R interval exceeding .20 seconds—called first degree heart block (or more properly auriculo-ventricular block)—the interval is reduced to or toward normal. (Fig. A)

Where the initial ventricular component of QRS complex is broadened beyond .10 seconds (the so-called bundle-branch block) there is no apparent change produced on E therapy. (Fig. C) In cases of the Wolff-Parkinson-White syndrome where this type of QRS is associated with a very short P-R interval, while there is no change in the curve; the patients have fewer and less severe attacks of paroxysmal tachycardia.

Abnormal S-T segments are most often seen (excluding digitalis effects, of course) in cases of coronary occlusion, coronary sclerosis and ventricular strain. In the first of these there is frequently a natural return to the base line without any treatment. In coronary sclerosis the administration of vitamin E is followed by a gradual return not commonly seen after any other medication. In ventricular strain one sees a prominent left or right axis-deviation followed by an S-T segment and T wave which is deviated to the opposite side of the base line from the maximum initial ventricular component. In these cases one commonly finds that the degree of axis-deviation is reduced on E therapy in a direction nearer the normal range.

T waves which are of less than normal height are increased in voltage, and those which are inverted are restored toward normal. (Figs. B, D, E, G, H, J). The T4 is quicker to respond than T1 and T3 (Figs. D, E, G).

Abnormal T waves in cases of ventricular strain are the slowest to respond, and sometimes do not show any improvement.

Not only uniform ventricular extra-systoles, but also the variform type, are reduced in number and frequency and often are abolished altogether.

There do not appear to be any more patients with auricular fibrillation who revert to sinus rhythm on E than on ordinary treatment. Of those who did, four dislodged emboli, of which two died. There have not been enough cases of flutter to draw any conclusions.

Discussion

The changes noted are somewhat comparable to those obtained in rabbits and rats by Martin and Faust.[1] They may be caused by better myocardial nutrition (Houchin and Mattill[2]), improved carbohydrate metabolism (Govier[3]), by the preservation of creatin (Pin[4]), or by a combination of all these effects.

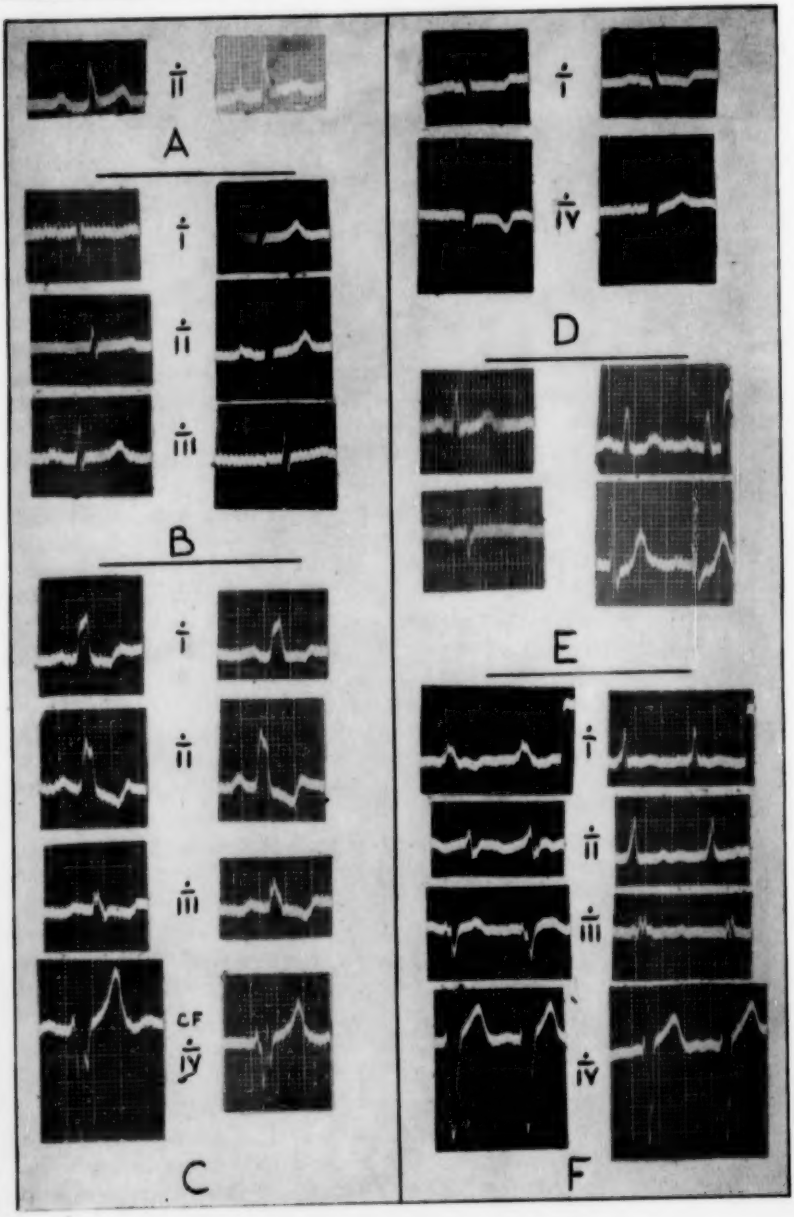
In myocardial infarction the normal paths of depolarization and polarization of the myocardium are disrupted. This process causes changes in the QRS complex, the RS-T segment and the T wave. It seems reasonable to assume that the electrocardiographic pattern will not return to normal unless the tissue which is involved has returned, in some manner, to its former state in so far as conductivity and contractility are concerned. This must imply an improvement in the blood supply to the ischemic tissue if the injury is recent, or revascularization of the scar tissue if the occlusion is of long standing. The first effect has been seen in various types of peripheral vascular disturbances under alpha tocopherol therapy[5, 6, 7]; the second has been seen in Dupuytren's Contracture,[8] in lupus erythematosus[9] and senile vulvovaginitis.[10]

In the author's experience there are cases where the changes caused by occlusion had remained unaltered over long periods of from six months to six years on conventional treatment but shortly after the vitamin E was administered had reverted to, or toward, normal.

This hypothesis could also apply to rheumatic heart disease which consists primarily of many small scars scattered among the muscle fibers.

A significant point in the treatment of cardiac patients with this vitamin is that electrocardiographic improvement lags weeks to months behind clinical improvement. The explanation is obvious from the pathological considerations above; and we have stressed this from the first.[5]

Whatever the mechanism by which these changes occur, the fact remains that many of the graphic disturbances indicative of myocardial damage revert toward normal after the patient has been given adequate doses of alpha tocopherol.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KEY TO FIGURES

Fig. A—Lead 2 showing P-R interval of .28 seconds. After 3½ weeks of 240 mg. alpha tocopherol daily lead 2 shows P-R interval of .20 sec.

Fig. F—Three limb leads of case of rheumatic heart disease with mitral stenosis. First leads show right axis-deviation with slightly inverted T1. After less than 4 weeks of 300 mg. daily same limb leads show normal range of axis-deviation and upright T1, as well as lower voltage in lead 3. This should suggest rotation.

Fig. C—Four standard leads showing common-type bundle-branch block eight months after occlusion. After 300 mg. daily for 2 months there is no change in QRS complexes except creation of RSR' S' complex in CF4.

Fig. D—Leads 1 and CF4 only. The first pair were obtained 5 years after an anterior occlusion and still show depressed ST1 and inverted T4. After only 2½ weeks on 300 mg. daily T4 became upright, but ST1 was still depressed.

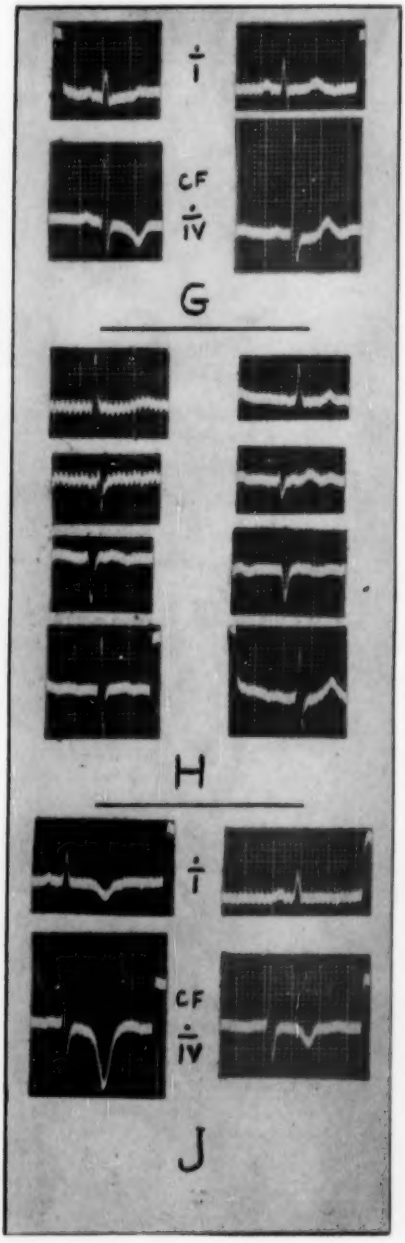
Fig. E—Leads 1 and CF4 only. Patient showed pulmonary edema and paroxysmal nocturnal dyspnea. BP 194/118. CF4 was QS type and T4 isoelectric. Digitalis was continued and 300 mg. alpha tocopherol added daily. After 2 months the second pair of curves was obtained showing no change in lead 1 but a high R4 and T4. BP reduced to 162/92.

Fig. F—Four standard leads, the first group taken 6 months after an anterior occlusion shows common type bundle-branch block, left axis-deviation and auricular fibrillation. After 4 months on 360 mg. daily, second group shows change in axis-deviation and reduction of QRS time from .12 sec. to .08 sec. (This last is very seldom seen in this series.)

Fig. G—Leads 1 and CF4 only. The first two were taken 2½ months after an anterior occlusion and show depressed ST1 and inverted T4. The second pair was obtained 1 month later and show normal lead 1 and T4 upright. The dose here was 240 mg. daily.

Fig. H—Hypertensive heart showing left ventricular strain. A dosage of 300 mg. daily for almost 4 months resulted in the second set which now showed less evidence of ventricular strain and an upright T4.

Fig. J—Leads 1 and CF4. Patient had had an occlusion over a year before the first pair were taken. After 270 mg. daily for exactly one month the second pair was taken. T1 is no longer inverted and T4 is much less so.



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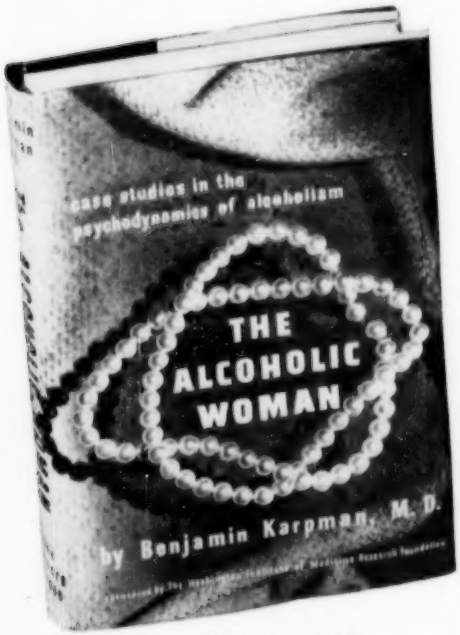
The Alcoholic Woman

by

BENJAMIN KARPMAN, M.D.



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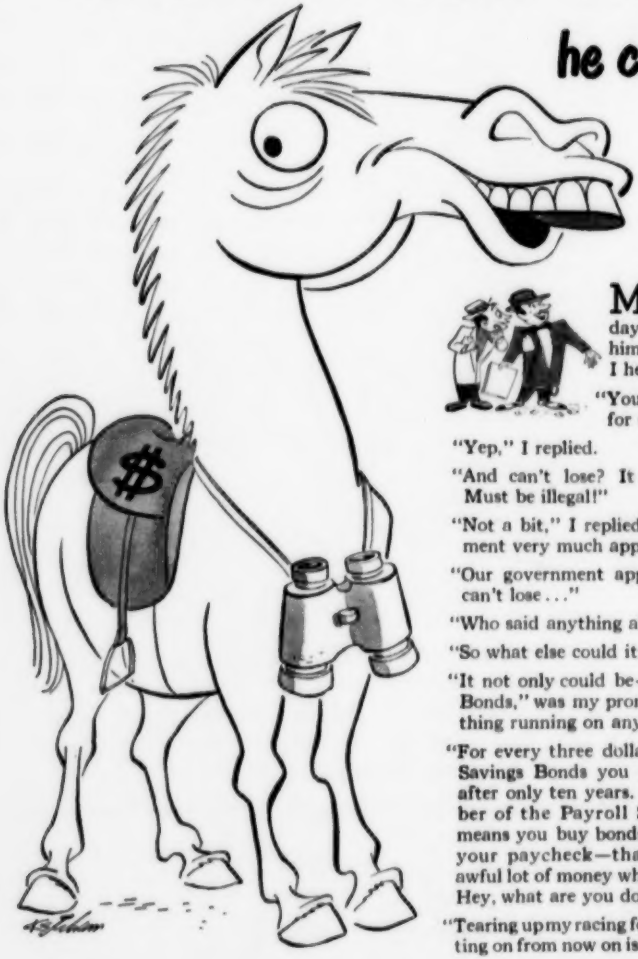

MY FRIEND and I were picking the ponies one day when I started telling him about *a sure thing* I heard about.

"You say it pays four bucks for every three?" he asked.

"Yep," I replied.

"And can't lose? It *automatically* wins? Must be illegal!"

"Not a bit," I replied. "In fact, the government very much approves..."

"Our government approves of a horse who can't lose..."

"Who said anything about a horse?" I asked.

"So what else could it be but a horse...?"

"It not only could be—but is—U. S. Savings Bonds," was my prompt reply. "The surest thing running on any track today.

"For every three dollars you invest in U. S. Savings Bonds you get four dollars back after only ten years. And if you're a member of the Payroll Savings Plan—which means you buy bonds *automatically* from your paycheck—that can amount to an awful lot of money when you're not looking. Hey, what are you doing?"

"Tearing up my racing form! The horse I'm betting on from now on is U. S. Savings Bonds."

Automatic saving is <u>sure</u> saving—U.S. Savings Bonds

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